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Atractaspis (Serpentes, Atractaspididae) the burrowing asp; a multidisciplinary minireview

E. KOCHVA
Department of Zoology, Tel Aviv University, Ramat Aviv, 69978 Tel Aviv, Israel

CONTENTS

SYNOPSIS. The family Atractaspididae is a highly modified derivative of a lineage that apparently arose early in the history of 'colubroid' snakes, and its taxonomy and relationship with other ophidian groups is still uncertain. Snakes of the genus *Atractaspis* have a characteristic venom apparatus, including the structure and function of the striking unit and of the venom glands. The composition of their venom is also unique in containing several low-molecular weight components, the sarafotoxins, which affect the cardiovascular system and are similar to the mammalian endothelins.

Dedication

This paper is dedicated to Dr. Garth Underwood on the occasion of the 35th anniversary of his classic 'Contribution to the Classification of Snakes' (1967), about which one may say:

This is a small book by a great man!

And also – (ברי"ר, ה, ז) *מכאן שהחזיק מועט את המרובה*

A little that contains a lot (Theodor & Albeck, 1996)

HISTORY

There are not many snake species that posed problems from the very beginning of their discovery; one of the most prominent ones is certainly that which we now call the genus *Atractaspis* of the family Atractaspididae (Fig. 1).

The first two specimens of *Atractaspis* were described by Reinhardt in 1843 as *Elaps irregularis*, a species that he considered to be extremely abnormal because of the presence of only a few, very small teeth. On the basis of squamation, Underwood inferred that at least one of the specimens was *A. dahomeyensis*. The genus *Atractaspis* was established by Smith in 1848 for the South African species *bibroni* and since then it was variously considered as a separate family, as a subfamily of the family Viperidae, and finally as a genus within the Viperinae.

Already Haas (1931), on the basis of the pattern of the head musculature, was unhappy with the inclusion of *Atractaspis* in the Viperidae, but it was not until 1961 that Monique Bourgeois, studying at the Université Officielle du Congo à Lubumbashi, came out with a challenging question: *Atractaspis* – a misfit among the Viperidae? This short note was followed by a detailed study suggesting the establishment of a separate subfamily for a group of opisthoglyphous colubrids together with *Atractaspis* (Bourgeois, 1968).

Underwood (1967) lists a long series of skeletal and other anatomical characters in which *Atractaspis* differs from the Viperidae and states: '*Atractaspis* differs so widely from the other vipers that I have no doubts about reviving a separate family group taxon to receive it' (p. 103). This he did after a detailed analysis that resulted in the resurrection of the subfamily Atractaspidinae (Gunther, 1858); and, finally, the establishment of a separate family, Atractaspididae, for the approximately 15 species of *Atractaspis* together with some African colubrid genera (Underwood & Kochva, 1993).

TAXONOMY

Recently several suggestions concerning the taxonomy and relationship of the *Atractaspis* species have been raised, mainly dealing with the question of which additional genera should be included in the family Atractaspididae and with which, if any, larger clades they should be grouped (Gravlund, 2001). Underwood himself (personal communication) is now reconsidering the composition of the family Atractaspididae in order to decide which genera, in addition to *Atractaspis*, should be included in it. However, no one is now questioning the separate status of the genus *Atractaspis*, and its apparent distinction from the other venomous snake families is widely, though not unanimously, agreed upon. *Atractaspis* thus certainly deserves the rank of a family of its own; this may also include some rear-fanged snakes that are apparently harmless as far as humans are concerned.

Fig. 1 *Atractaspis engaddensis* in a combined defensive/offensive position. Note the arched neck and the beginning of the coiled body with exposed tip of the tail.

DISTRIBUTION

The distribution of the *Atractaspis* species is unique (Fig. 2), starting from the Cape of South Africa, through the entire breadth of central Africa and along the Rift Valley to Arabia, Sinai, Jordan and Israel, reaching its northernmost border at Mount Gilboa (Al-Oran & Amr, 1995; Kochva, 1998).

It is in Israel that the last species of *Atractaspis* was found and described. It was first recorded by Aharoni in 1945 as *Atractaspis aterrima* and later described as a new species, *Atractaspis engaddensis*, by Haas in 1950. *A. engaddensis* is very similar to the Arabian *A. microlepidota andersoni* (Gasperetti, 1988), but a decision on the exact status of the two will have to await further information on the distribution of *Atractaspis* forms in the Arabian Peninsula as well as on the toxicity and composition of their venoms (see also Al-Sadoon *et al.*, 1991; Al-Sadoon & Abdo, 1991; Schätti & Gasperetti, 1994).

BEHAVIOUR

Species of the genus *Atractaspis* are desert-dwelling, fossorial snakes, whose behaviour and natural history are not well known. The Israeli species, *A. engaddensis,* is mostly found in the Negev desert and Dead Sea area, but it also extends to the Judean desert and along the Jordan Valley up to Mount Gilboa (Fig. 2).

A. engaddensis feeds mainly on skinks, but also on lizards and geckoes that are caught at night above or below ground, beneath stones or other objects. In captivity, it also accepts baby mice and rats. *A. microlepidota* feeds on other snakes such *Typhlops* and *Leptotyphlops*, amphibians and small mammals, mostly rodents (Scortecci, 1939; Greene, 1997). In a four-year field study carried out by Akani *et al.* (2001) in south-eastern Nigeria, it was found that *A. irregularis* fed mainly on rodents, while *A. aterrima* and *A. corpulenta* ate lizards, skinks and snakes.

The swallowing behaviour of *Atractaspis* may be influenced by its nearly vestigial teeth. As described for *A. bibroni,* it is characterised by a rather inefficient transport mechanism in which the snake forces its head over the prey with lateral rotations around a vertical axis, rather than with the 'pterygoid walk' used by other snakes. This can be considered to be an adaptation towards feeding in narrow spaces and explained by the lack of connection between the pterygoid and palatine bones that are separated by a wide gap bridged by a ligament (Deufel & Cundall, 2000; MS; see also Underwood and Kochva, 1993).

A. engaddensis lays 2–3 elongated eggs during the months of September–November and hatching occurs after about 3 months (Fig. 3).

An interesting behavioural feature of this snake is its threat posture during which it presses its head to the ground while arching its neck (Fig. 1). This may turn either into a strike or into what appears to be a defensive display mechanism (Greene, 1979; 1997; Golani & Kochva, 1993). The snake forms a tight coil with the head hidden underneath the body and the wriggling tail is exposed above

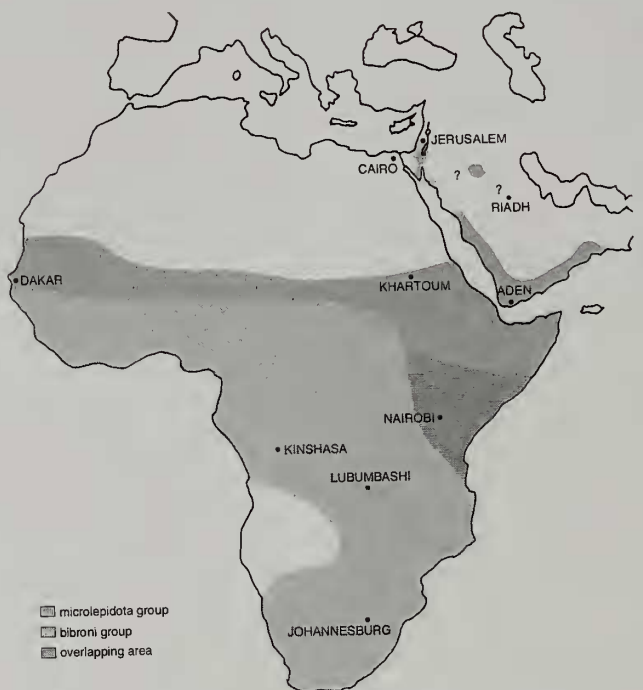

Fig. 2 Distribution map of *Atractaspis* species with the southern African *bibroni* group and the northern *microlepidota* group reaching the region of Mount Gilboa (after Underwood & Kochva, 1993; Joger, 1997).

Fig. 3 Hatching of *Atractaspis engaddensis*.

Fig. 4 Defense mimicry posture by *Atractaspis engaddensis*, with hidden head and exposed tail.

Fig. 5 Tail poking by *Atractaspis engaddensis*.

the coil so as to mimic the head (Fig. 4). The tail ends in a sharp tip that the otherwise immobile snake uses for poking when grasped at the posterior end of the body (Fig. 5). This behaviour may be mistaken for a genuine strike with the fang and deter any potential predator. Should that not suffice, there is always the hidden head that can be produced quickly from underneath the body coils and inflict a real, painful and dangerous strike.

VENOM APPARATUS

The venom apparatus of *Atractaspis* has not been dealt with in great detail beyond the general statement that the maxillary/fang unit is similar to that of vipers. This similarity is, however, superficial as the articulation between the prefrontal and maxilla in *Atractaspis* is in the form of a ball and socket articulation, which is more restricted in its movements, but apparently stronger (Pasqual, 1962). This condition may be important for the peculiar striking of these snakes, which is performed with one fang at a time while the mouth remains almost entirely closed (Fig. 6). Striking in this manner may be considered as a special adaptation for fossorial snakes that feed in narrow burrows underground.

The venom fangs are relatively long and canaliculate and possess a blade-like ridge near the orifice of the fang (Kochva & Meier, 1986), which may increase the wound and cause additional tissue damage during the strike, thus facilitating the spread of venom. Analyses of films taken during a strike through plastic sheathings show first the establishment of a firm contact of the head with the substrate, followed by the erection of the fang and piercing of the substrate by arching, lateral bending and downward rotation of the head (Fig. 6). Ejection of the venom is performed while the fang moves backward, further cutting through the surface (Golani & Kochva, 1988).

The venom glands have a distinctive structure with secretion tubules arranged concentrically around the main lumen (Fig. 7). Unlike the viperids and elapids, there are no differentiated mucous accessory glands, but mucous cells are found in each of the secretion tubules close to the central lumen (Kochva *et al.*, 1967). As in the other families of venomous snakes, there are species (the *microlepidota* group, Underwood & Kochva, 1993) with elongated venom glands that reach far beyond the corner of the mouth (Kochva, 1959). The compressor muscle accompanies the gland along its entire length and probably squeezes it during the strike so as to increase the pressure in the central lumen and push the venom through the venom duct, fang canal and into the wound. The species with short glands (the *bibroni* group) have a short, but thicker compressor.

In a 756 mm long *A. engaddensis* the right gland reached the 30th ventral and was 70 mm long, while in *A. microlepidota* it may reach one third of the body length – more than 300 mm in a specimen of 900 mm (Scortecci, 1939). The left gland is usually longer than the right gland in both species and it is sometimes twisted along its longitudinal axis (Fig. 8).

VENOM

The venom of *Atractaspis* remained unknown for a long time, probably because not many serious bites were reported until now and it was thus ignored by toxinologists. In addition, the venom is very difficult to obtain not only because of the relative paucity of specimens collected, but also because of the difficulty in extracting

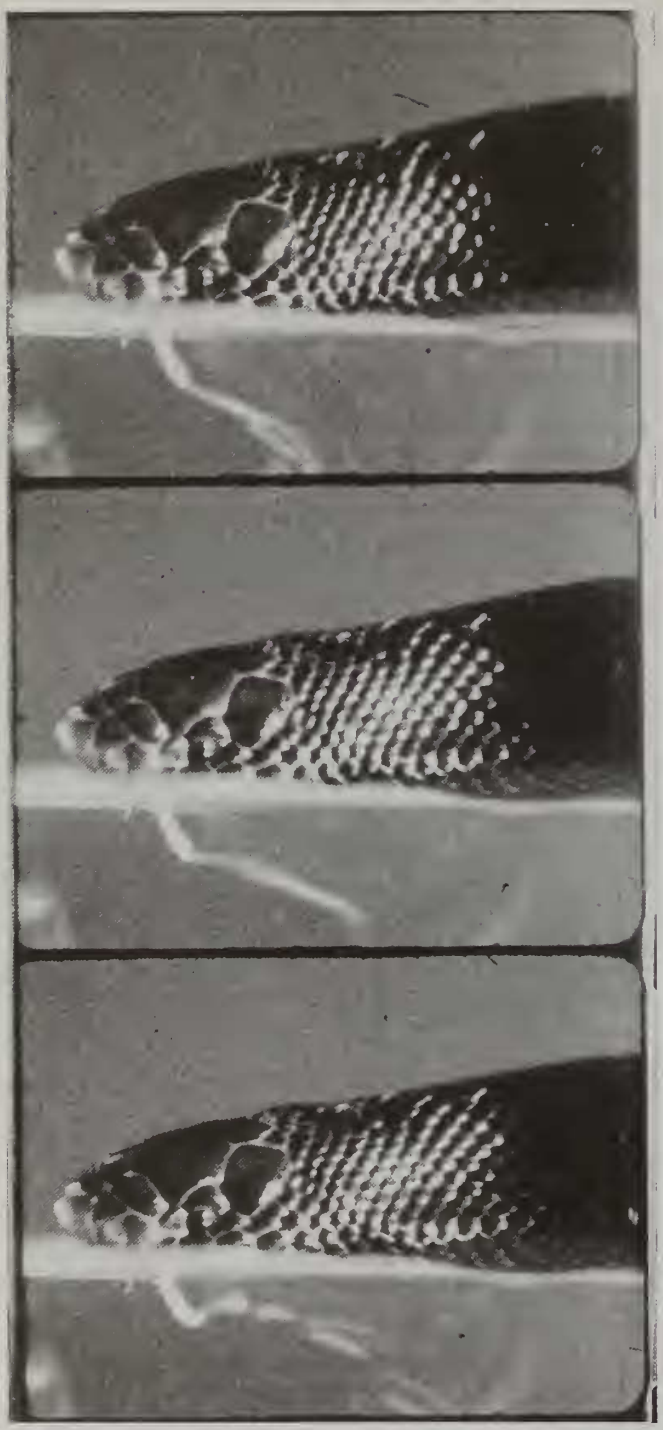

Fig. 6 Striking sequence by *Atractaspis engaddensis*; sequence from film (see text).

it from the glands. *Atractaspis,* with its spade-shaped head, cannot be milked the way other venomous snakes are; a special method had to be devised as shown in Figure 9.

Even today the biochemistry and pharmacology of the venom are known for only a few species, with almost all the information available originating from research with the venom of *A. engaddensis*.

The toxicity of the venom varies among the species tested, the most potent venom being that of *A. engaddensis*, exceeding by 40 times or more that of some other species (Table 1). It contains a set

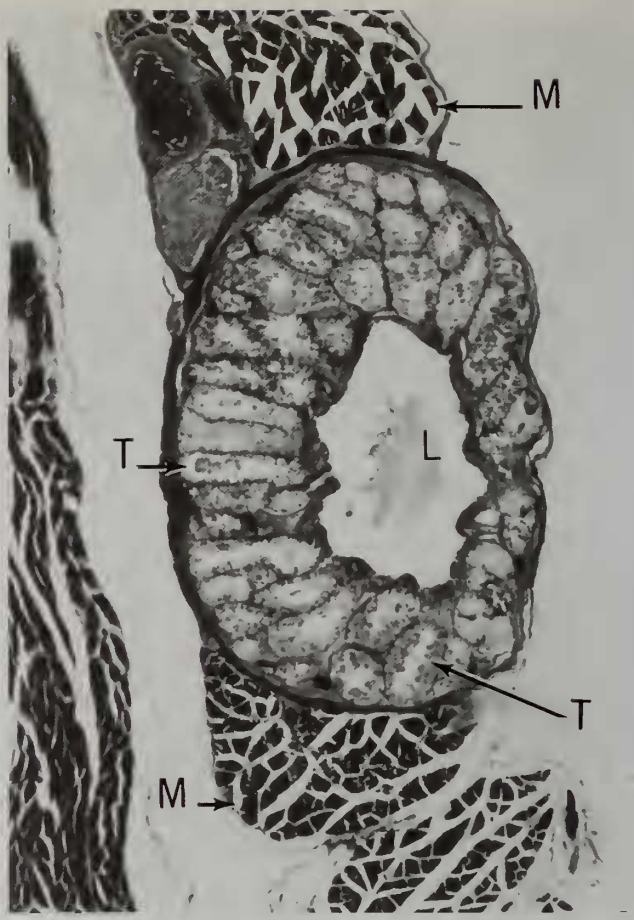

Fig. 7 Venom gland of *Atractaspis engaddensis*, cross section: M = compressor muscle, L = lumen of venom gland, T = secretion tubules.

of enzymes not unlike those of other venomous snakes, a quite powerful hemorrhagin and a group of low-molecular weight toxins, the sarafotoxins, named after the Hebrew name of *A. engaddensis* – Saraf. When the venom was first fractionated by molecular sieving on a Sephadex G-50 column, 7 protein peaks were obtained. The first two contained high-molecular weight proteins with a hemorrhagic factor and the enzyme L-amino acid oxidase; the third peak showed phospholipase A_2 activity and peaks 5 and 6 contained very low molecular weight peptides, which made up 40% of the venom proteins and were highly toxic in i.v.-injected mice (Kochva *et al.*, 1982). These fractions were further purified resulting in several toxins characterised as sarafotoxins (SRTX), which showed

Table 1 Toxicity of *Atractaspis* venoms, sarafotoxins, and mammalian endothelins in mice, LD_{50} (ng/g b.w.)

A. bibroni	500
A. dahomeyensis	2000
A. microlepidota	>2000
A. micropholis	>3000
A. engaddensis	75
Sarafotoxin-a	10
Sarafotoxin-b	10
Sarafotoxin-c	300
Sarafotoxin-d/e	>2000
Endothelin-1	15
Endothelin-3	30

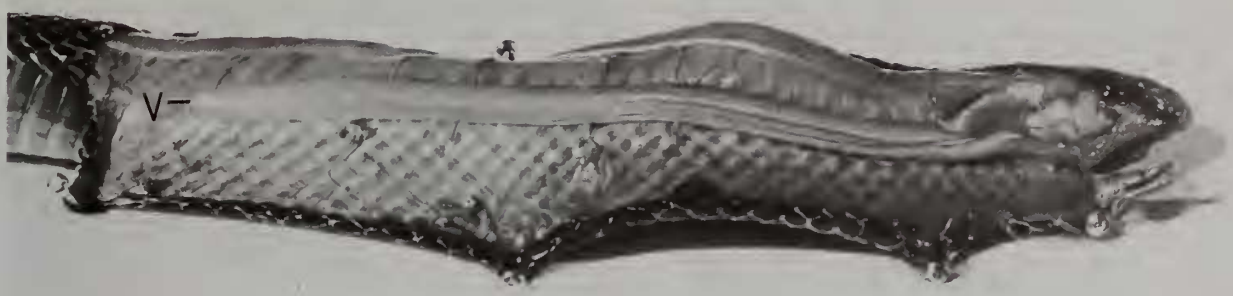

Fig. 8 Elongated venom glands of *Atractaspis engaddensis*: V = venom gland.

Fig. 9 Venom extraction from *Atractaspis engaddensis* using a piece of tubing for safety and a parafilm-covered lid for the collection of venom.

a high degree of structural homology amongst themselves and with a group of active peptides that were isolated from mammalian endothelium, the endothelins (Fig. 10). The sarafotoxins and endothelins are also similar in their pharmacological activity and are composed of 21 amino acid residues, with two disulphide bridges between Cys 1–15 and 3–11 (Yanagisawa *et al.*, 1988; Takasaki *et al.*, 1988; Wollberg *et al.*, 1990; Kochva *et al.*, 1993). Another member of the sarafotoxin/endothelin family, bibrotoxin, was isolated from the venom of *A. bibroni*. It differs from SRTX-b in only one amino acid substitution and induces vasoconstriction in rat aorta (Becker *et al.*, 1993). The venom of *A. m. microlepidota* contains a series of peptides with a somewhat higher molecular weight that are composed of 24 amino acids (Ducancel *et al.*, 1999) and are apparently less toxic than SRTX-a or b.

The sarafotoxins and endothelins are now synthesised by pharmaceutical companies and are widely used in both basic and applied research, both clinical and industrial, in the field of cardiology and in blood pressure studies (Ducancel *et al.*, 1999; Yaakov *et al.*, 2000).

The various sarafotoxins (and endothelins) differ in their activity and toxicity, the most potent ones being SRTX-a and SRTX-b (Table 1), which exert a strong influence on the cardiovascular system (Wollberg *et al.*, 1988). SRTX-b shows three, apparently separate, effects on the heart: 1) positive inotropicity, which is manifested by an increased contractility in isolated hearts and heart muscles and in *in vivo* injected mice with sublethal doses of the

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 1                5                  10                  15                  20
Cys-Ser-Cys-Lys-Asp-Met-Thr-Asp-Lys-Glu-Cys-Leu-Asn-Phe-Cys-His-Gln-Asp-Val-Ile-Trp    SRTX-a
Cys  *  Cys  *  *  *  Ser  *  *  *  Cys  *  *  *  Cys  *  *  *  *  *  *    SRTX-a1
Cys  *  Cys  *  *  *  *  *  *  *  Cys  *  Tyr  *  Cys  *  *  *  *  *  *    SRTX-b
Cys  *  Cys  *  *  *  Ser  *  *  *  Cys  *  Tyr  *  Cys  *  *  *  *  *  *    SRTX-b1
Cys  *  Cys-Ala  *  *  *  *  *  *  Cys  *  Tyr  *  Cys  *  *  *  *  *  *    BTX
Cys-Thr-Cys-Asn  *  *  *  *  Glu  *  Cys  *  *  *  Cys  *  *  *  *  *  *    SRTX-c
Cys-Thr-Cys  *  *  *  *  *  *  *  Cys  *  Tyr  *  Cys  *  *  Gly-Ile  *  *    SRTX-d/e
Cys  *  Cys-Asn  * Ile-Asn  *  *  *  Cys Met Tyr  *  Cys  *  *  *  *  *  * Asp-Glu-Pro A. microlepidota
Cys  *  Cys-Ser-Ser-Leu-Met  *  *  *  Cys-Val-Tyr  *  Cys  *  Leu  *  Ile  *  *    ET-1
Cys  *  Cys-Ser-Ser-Trp-Leu  *  *  *  Cys-Val-Tyr  *  Cys  *  Leu  *  Ile  *  *    ET-2
Cys  *  Cys-Asn-Ser-Trp-Leu  *  *  *  Cys-Val-Tyr  *  Cys  *  Leu  *  Ile  *  *    VIC
Cys-Thr-Cys-Phe-Thr-Tyr-Lys  *  *  *  Cys-Val-Tyr-Tyr-Cys  *  Leu  *  Ile  *  *    ET-3
Cys  *  Cys-Ala-Thr-Phe-Leu  *  *  *  Cys-Val-Tyr  *  Cys  *  Leu  *  Ile  *  *    ET-trout (1999)
 1                5                  10                  15                  20
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Fig. 10 Amino acid sequences of sarafotoxins and endothelins: BTX = bibrotoxin, ET = endothelin, SRTX = sarafotoxin, VIC = vasoactive intestinal contractor.

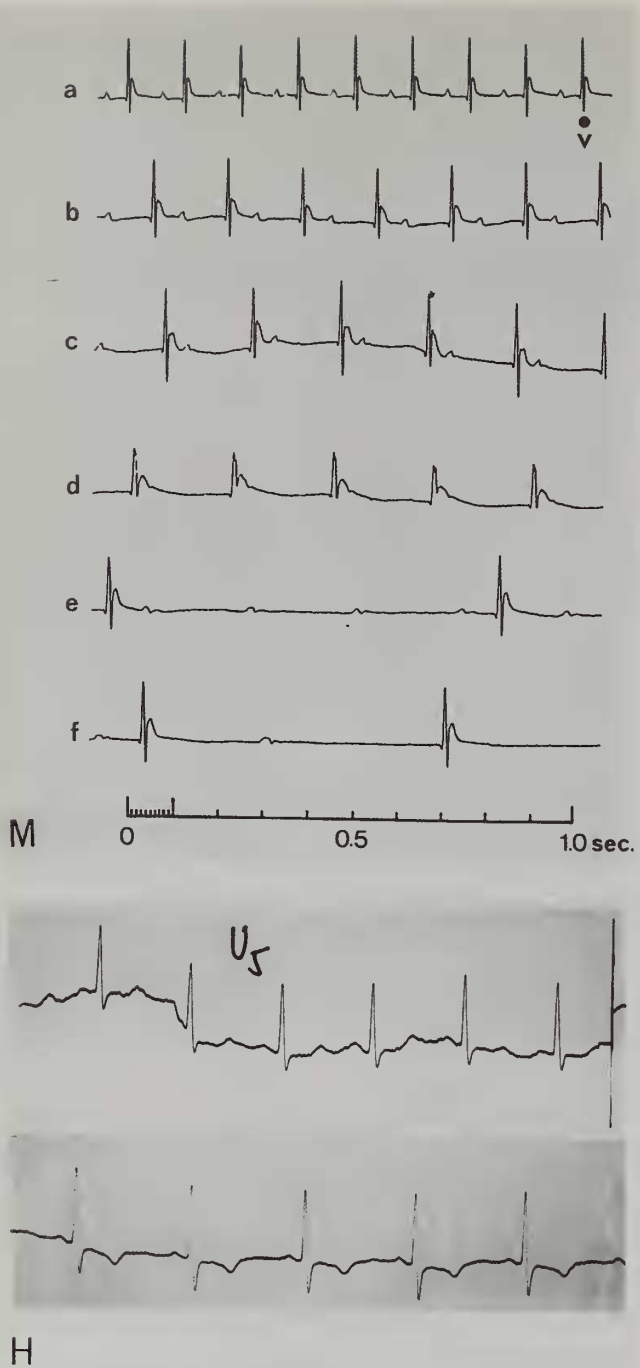

Fig. 11 ECG recording after *Atractaspis engaddensis* envenomation. M = mouse: v = venom injection; b – f: 120 – 600 seconds after venom injection. H = human: upper trace – at admission to the hospital; lower trace – 24 hours after the bite (see text).

toxin; 2) direct effect of the toxin on the cardiac conducting system; and 3) cardiac ischemia, which is caused by constriction of the coronary blood vessels. The latter two cause severe A–V block, which may lead to cardiac arrest. The cardiotoxic effects are manifested by marked changes in the ECG, in both human victims and in mice injected with either SRTX-b or whole venom (Fig. 11). These changes include an increase in amplitude of the R- and T-waves, a prolongation of the P–R interval, 'dropped beats' and complete A–V block and cardiac arrest. In addition, SRTX-b causes contraction of other blood vessels and may be considered as one of the most potent vasoconstrictors.

In human patients too, cardiotoxic symptoms and a rise in blood pressure were observed, but were considered as secondary developments of some kind of neurotoxic effects. However, neither presynaptic nor postsynaptic neurotoxicity was observed in laboratory tests with nerve-muscle preparations using whole *A. engaddensis* venom (Weiser *et al.*, 1984); SRTX does show specific binding to different isolated regions of brain preparations, with the highest binding capacity found in the cerebellum, choroid plexus and hippocampus (Ambar *et al.*, 1988), but its function there is not known.

In human bites by *A. engaddensis* and *A. irregularis*, some of which were extremely severe, changes in the ECG were observed (Fig. 11), including S–T elevation or depression, flattening of the T-waves and prolonged P–R intervals pointing to myocardial ischemia and atrioventricular conduction abnormalities (Chajek *et al.*, 1974; Warrell, 1995; Kurnik, *et al.*, 1999). The transient atrioventricular block that developed in a 17-year old boy bitten on his left foot was considered to be a secondary complication of the bite (Alkan and Sukenik, 1974), rather than a direct influence of the toxins on the heart.

The other systemic symptoms, which may develop within minutes, include fever, nausea, general weakness, sweating, pallor, fluctuations in the level of consciousness and a rise in blood pressure (Doucet & Lepesme, 1953; Chajek *et al.*, 1974; Kurnik *et al.*, 1999).

Most bites were on the fingers and the local effects were demonstrated mainly by gross oedema of the hand that extended up to the forearm and shoulder (*A. irregularis* – Doucet and Lepesme, 1953; *A. corpulenta* – Gunders *et al.*, 1960; *A. microlepidota* – Warrell *et al.*, 1976) and by blistering and serous vesicles that appeared at the site of the bite and underwent hemorrhagic transformation (Fig. 12; Kurnik *et al.*, 1999). In some previously reported cases (Chajek *et al.*, 1974; Chajek & Gunders, 1977), local necrosis developed that required surgical intervention including amputation. In two cases, one by *A. bibroni*, the other by *A. engaddensis*, the bitten finger partially or fully recovered within a month, but tenderness of the bitten site remained for a long time (Stewart, 1965; Kurnik *et al.*, 1999).

Although the bites by several species of *Atractaspis*, such as *A. dahomeyensis, A. aterrima, A. corpulenta* and *A. bibroni* were mild (Warrell *et al.*, 1976; Tilbury & Branch, 1989), *A. engaddensis, A. irregularis* and perhaps other species should be regarded as dangerous

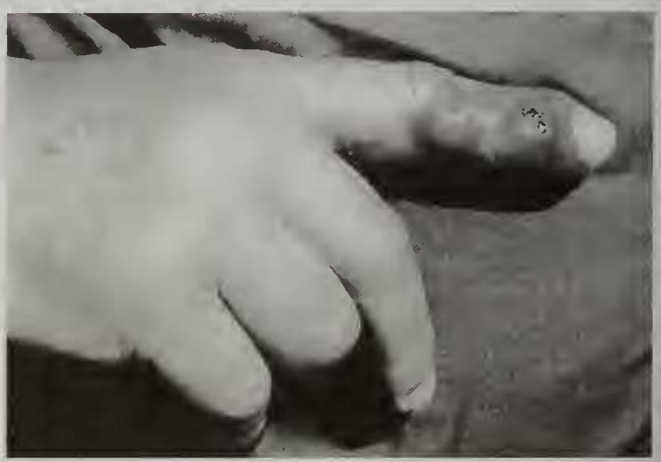

Fig. 12 Bitten index finger showing hemorrhagic transformation of serous vesicles.

Fig. 13 Ridges on the teeth of *Pachyrhachis problematicus* (arrow).

mainly because of their influence on the cardiovascular system, which may lead to death. Only a very small number of lethal cases has been recorded until now, perhaps a total of five, three by *A. microlepidota* (one adult man and two girls aged 4 and 6), one by *A. irregularis*, an adult man, and one unknown (Corkill *et al.*, 1959; Warrell *et al.*, 1976). Despite the fact that *A. engaddensis* has one of the most potent venoms known, all patients bitten by this species finally recovered, one probably due to 'the immediate and energetic treatment he received' (Chajek *et al.*, 1974). Most recently (July, 2002) a forty-six-year-old man was bitten on the inner aspect of the right thumb while trying to catch an *Atractaspis engaddensis* near his home in the Judean Desert, some 15 km north west of Jericho. He was taken to the hospital where he arrived after about 40 minutes in serious condition. Resuscitation failed and he was pronounced dead after about 45 minutes (Nadir & Stalnikowicz, personal communication). This is the first death by an *Atractaspis engaddensis* bite in Israel. Another recent case, from Saudi Arabia, involved a two-year-old-girl who died within one hour after being bitten on the foot by what was identified as *A. microlepidota engaddensis*. The region where the bite occurred, at Diriyah near Riyadh, Saudi Arabia, is a new distribution record for *engaddensis* (Al-Sadoon & Abdo, 1991; see also Al-Sadoon *et al.*, 1991; Gasperetti, 1988l; Joger, 1997; Schätti & Gasperetti, 1994).

It should be pointed out that the toxicity of the venom of certain species, such as *A. microlepidota,* may vary according to distribution, causing death in certain cases (see above) or containing less potent toxins in others (above and Table 1).

As with other venoms, snakes and some mammals are also resistant to *Atractaspis engaddensis* venom, including the local mongoose (*Herpestes ichneumon,* Bdolah *et al.*, 1997). At least in one instance, it was found that a mongoose (*Paracynictis selousi)* fed on a specimen of *A. bibroni* (Greene, 1997).

There is no antiserum available against any of the *Atractaspis* species.

EVOLUTION

The discussion of snake origin and evolution has been recently revived by a new and renewed examination and analysis of the fossils discovered by Haas (1979; 1980a; 1980b) at an Upper Cretaceous site north of Jerusalem. While the debate on the ecological origin (marine or terrestrial) and the relationships of these specimens (mosasauroid or macrostomatan) is still going on (Lee & Caldwell, 1998; Greene & Cundall, 2000; Tchernov *et al.*, 2000), Rieppel & Zaher (2001) have recently concluded that '*Pachyrhachis* is neither a basal snake, nor a link between snakes and mosasauroids, but shows macrostomatan affinities instead'. *Pachyrhachis* possesses ridges or cutting edges on its teeth (Rieppel & Kearney, 2001; Fig. 13) and the teeth of another fossil, *Haasiophis,* have still to be further investigated in detail. Should furrows or any other suggestive structures be found, they could be taken as plausible signs for the existence of some kind of glands that might have secreted active substances, even before the appearance of caenophidian snakes.

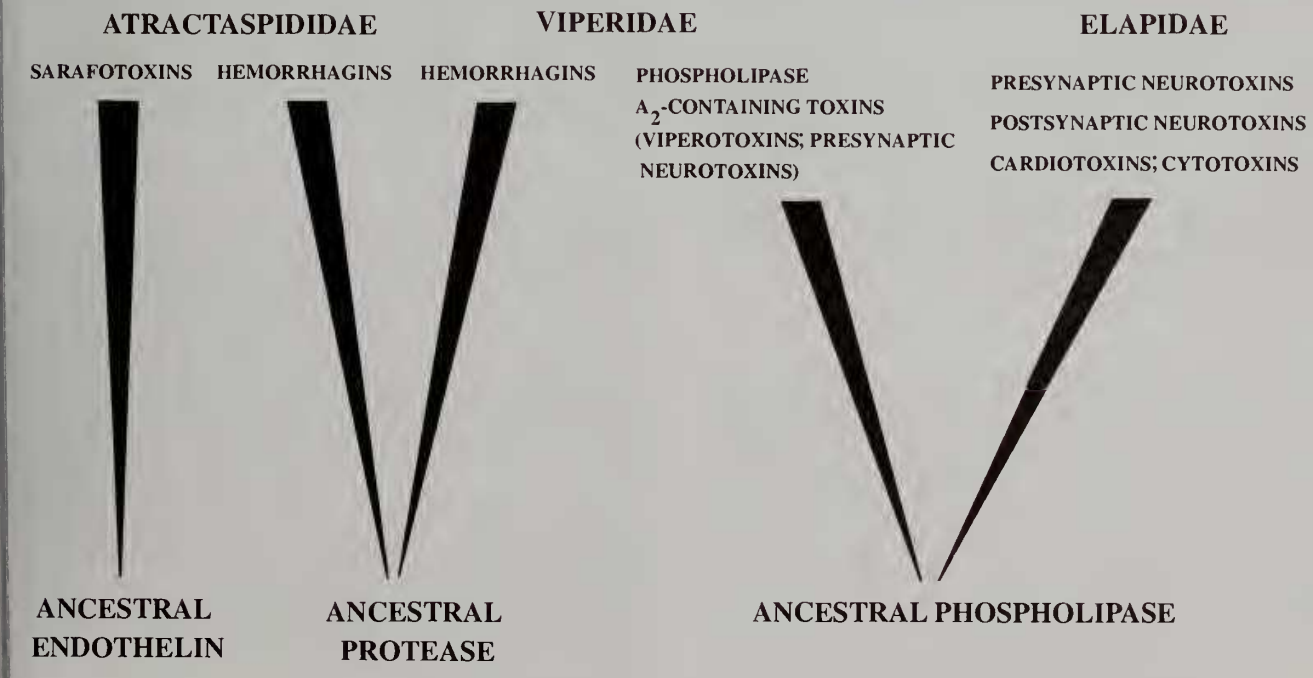

Fig. 14 Schematic representation of the possible origin of some major snake venom toxins from enzymatic precursors (partly after Strydom, 1979).

It has been suggested that a system that produced active substances with the means of introducing them into the prey probably lay at the foundation of the major radiations of higher snakes (Underwood & Kochva, 1993). This system underwent further evolution in the Atractaspididae (mainly *Atractaspis*), Viperidae, Elapidae and several lineages of 'Colubridae'.

Some of the active substances were probably enzymatic in nature and related to enzymes secreted by evolutionarily 'older' glands, such as the pancreas. Indeed, phospholipases found in the venom of Elapidae, for instance, show sequence homology with the enzymes secreted by the mammalian pancreas. Some of the ancestral enzymes developed into toxins, such as hemorrhagins and neurotoxins, with or without loss of enzymatic activity (Fig. 14; Strydom, 1979; Kochva, 1987).

Hemorrhagins are found in two families (Viperidae and Atractaspididae); presynaptic neurotoxins in two (Elapidae and Viperidae); and two families each possess a specific and unique group of toxins – postsynaptic neurotoxins in elapids and sarafotoxins in *Atractaspis*.

The hemorrhagin found in the venom of *Atractaspis* is neutralised by antibodies against *Vipera palaestinae* venom (Ovadia, 1987) and may thus be related to viperid hemorrhagins, originating from some kind or kinds of protease. The presynaptic and postsynaptic neurotoxins, as well as the cytotoxins and cardiotoxins, apparently originate from phospholipase-like molecules. The enzyme phospholipase A_2 may be part of the presynaptic neurotoxins and its enzymatic activity may still be essential for its toxicity. The postsynaptic neurotoxins, the cytotoxins and the cardiotoxins apparently underwent major changes including loss of enzymatic activity, chain shortening and gain of neurotoxicity (Strydom, 1979).

The sarafotoxins are structurally very similar to the endothelins, which are evolutionarily highly conserved, and are found in all vertebrates, as well as in some invertebrate groups. It should be emphasised, however, that the genes of the mammalian endothelins were found on three separate chromosomes, whereas the sarafotoxin genes seem to be located on the same chromosome. The organisation of the SRTX genes of both *A. engaddensis* and *A. m. microlepidota* and their precursors are also different from those of the endothelins and may have evolved separately (Ducancel *et al.*, 1993; 1999).

There is, of course, a great deal of information still missing, but the evolution of the sarafotoxins and of some of the other snake venom toxins and their use in feeding and defense may best be defined as exaptations; these are features that once had different functions but are now used in a new role that enhances the fitness of their bearers (Gould & Vrba, 1982).

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I am very much indebted to my co-workers in Israel, South Africa and Japan for their major share in the different disciplines of the *Atractaspis* research and to the undergraduate and graduate students (listed in the references) and to many more who picked up the sarafotoxin project (with or without endothelin) and developed it into such a broad and deeply interesting field, with still much more to be expected in the future.

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